Clinical Optometry

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REVIEW

From the Ocular Surface to Neurophysiology: An Integrative Review of Digital Eye Strain

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Video abstract

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Abstract: Digital eye strain is a cross-system condition that arises from interactions between visual physiology and digital displays. As the use of screens has grown in work, education, and everyday life, researchers have sought to describe this condition through symptom questionnaires (CVS-Q, CVSS17, DESQ), measurements of the ocular surface and blinking, tests of accommodation and vergence, and, in related visual fatigue studies, neurophysiological methods such as electromyography and electroencephalography (EEG). Nevertheless, these methods have usually examined individual mechanisms separately. Interventions—ranging from lubricating the ocular surface and filtering blue light to ergonomic changes, task-specific refractive correction, and scheduled micro-breaks—have seldom been guided by a unified mechanistic framework. This review compiles evidence from 128 studies to identify six mechanistic domains relevant for clinical assessment. These include ocular surface and blink dynamics, accommodation and vergence, musculoskeletal load, device-related optical stress, cognitive load, and neural markers. A systematic search of PubMed/MEDLINE, Embase, Scopus, and Web of Science was carried out through September 30th, 2025, in accordance with PRISMA guidelines. Eligible studies included randomized trials, controlled interventions, cohort studies, and laboratory experiments, with methodological quality evaluated using the Mixed Methods Appraisal Tool. Results across pediatric, adolescent, working-age, and presbyopic groups indicate that cognitive load reduces blinking and destabilizes accommodation. Binocular incongruence links visual effort with trapezius activation, and EEG markers can precede subjective fatigue. Interventions aligned with these mechanisms—such as ocular surface care, accommodative/vergence correction, ergonomic optimization, and micro-break scheduling—are supported across all domains. The review proposes a mechanism-based triage model to assist clinicians in prioritizing interventions based on the patient's exposure profile, breaking neuro-ocular feedback loops, and preserving functional performance in individuals exposed to screens. The framework provides an actionable, mechanism-first triage checklist for optometric practice.

Plain Language Summary: Digital eye strain is the collection of eye, neck, and mental fatigue symptoms that many people experience when using computers, smartphones, or tablets for long periods. These symptoms include dryness, burning, blurred vision, headaches, and shoulder or neck discomfort. Although common, digital eye strain does not come from a single cause. Instead, it results from several interacting factors involving the eyes, brain, posture, and design of digital screens.

This review synthesizes 128 studies to explain the development of digital eye strain and how it can be more effectively managed. Reduced blink frequency and completeness due to screen use destabilize the tear film, leading to dryness. Near focus over an extended period can cause strain on accommodation and vergence systems, particularly among children, teenagers, and those with untreated vision impairments. Poor posture, short viewing distances, and small screens increase the activity of neck and shoulder muscles. Performing multiple tasks simultaneously or engaging in demanding digital work significantly increases mental stress by requiring greater mental effort and resulting in a lower blink rate. Studies using EEG demonstrate brain fatigue preceding the onset of symptoms.

The review proposes a practical, mechanism-based approach for clinicians: identify which system is most stressed (ocular surface, focusing, posture, device factors, or cognitive load) and match treatment accordingly. This may include lubricating drops, blink training, vision therapy, ergonomic adjustments, scheduled movement breaks, or screen-based lighting changes. Understanding these mechanisms can help people use digital devices more comfortably and protect long-term visual performance.

https://doi.org/10.2147/OPTO.S586750
Received: 16 December 2025
Accepted: 17 February 2026
Published: 3 March 2026

Keywords: digital eye strain, computer vision syndrome, ocular surface, electroencephalography, ergonomics, musculoskeletal disorder

Introduction

Digital eye strain, also known as computer vision syndrome, encompasses a spectrum of ocular, musculoskeletal, and neurocognitive symptoms arising from sustained use of digital devices.[1] As digital screens are embedded in professional, educational, and recreational settings, digital eye strain has become a global public health concern across all age groups. Symptoms include ocular dryness, irritation, blurred vision, diplopia, headache, photophobia, musculoskeletal discomfort, and sleep disruption. These manifestations reflect the multifactorial nature of the digital eye strain, involving ocular surface instability, accommodative and vergence stress, postural strain, device-related optical stress, and cognitive overload.[1,2]

Digital eye strain is relevant from an ophthalmic or optometric standpoint because it directly affects ocular physiology, binocular vision, and accommodative mechanisms. Questionnaires such as the Computer Vision Syndrome Questionnaire (CVS-Q),[3] the Computer-Related Visual and Ocular Symptoms Scale (CVSS17),[4] and the Digital Eye Strain Questionnaire (DESQ),[5] serve as reliable instruments for evaluating symptoms in both occupational and clinical settings. However, these instruments capture primarily peripheral ocular symptoms and often do not account for central fatigue and attentional modulation. Research has shown that digital activities can reduce blinking rate and disrupt the tear film,[6–8] and smartphone and tablet users often experience difficulties with accommodative and vergence dysfunctions.[9–11] Musculoskeletal surveys highlight the high prevalence of neck and shoulder pain among students and professionals.[12–14] At the same time, device-related optical stressors, such as flickers and blue-enriched light, further exacerbate digital eye strain.[15,16]

In addition to ocular and ergonomic mechanisms, cognitive load has appeared as a critical determinant of digital eye strain. Studies have shown that greater task complexity amplifies accommodative microfluctuations and intensifies symptom severity.[1,17] Neurophysiological investigations using electroencephalography (EEG) reveal increased frontal theta activity, reduced occipital alpha power, and altered connectivity during digital tasks,[18,19] highlighting the cerebral basis of digital eye strain and offering objective markers of central fatigue. The most recent experimental results demonstrate that cognitive demand combined with short viewing distances amplifies ocular suppression and musculoskeletal load, highlighting digital eye strain as a cross-system phenomenon requiring integrative management.[20]

Despite this growing body of evidence, previous reviews have often emphasized isolated mechanisms—such as ocular surface instability, ergonomic strain, or broad prevalence estimates—without integrating ocular physiology, ergonomics, and neurophysiology into a unified framework, thereby limiting clinicians' ability to identify dominant drivers and prioritize targeted interventions in practice.[21–23] Taking an optometric perspective, the study hypothesizes that a focused, mechanism-based pathway combining targeted peripheral tests such as tear film stability, blink dynamics, and accommodative vergence measures with selective neurophysiological adjuncts (such as EEG markers of cognitive fatigue and accommodative microfluctuations), can identify treatable drivers in most digital eye strain patients and improve both symptoms and functional outcomes when applied within a staged management algorithm. This review uniquely contributes by providing an integrative framework that bridges ocular physiology with cognitive and ergonomic stressors in digital eye strain, proposing a mechanism-based triage method to guide clinicians in prioritizing interventions, and identifying potential research directions that unify experimental and clinical evidence. Therefore, this review aims to translate evidence from original clinical and experimental studies into a practical optometric-centered framework for the assessment and management of digital eye strain, enabling mechanism-based triage, guiding targeted treatment strategies, and showing prioritized directions for future research.

Methods

Search Strategy and Selection Criteria

A broad literature review was completed to summarize clinical, experimental, epidemiological, and neurophysiological evidence on digital eye strain. A single reviewer searched PubMed/MEDLINE, Embase, Scopus, and Web of Science from the start of each database until September 30th, 2025. The search used keywords and MeSH/Emtree terms like "digital eye strain", "computer vision syndrome", "blink rate", "tear film stability", "accommodation", "vergence", "flicker", "blue light", "EEG", and "visual fatigue". Search strings combined exposure terms: visual display terminal (VDT), smartphone, tablet, virtual reality (VR)/augmented reality (AR), outcomes (asthenopia, dry eye, accommodative microfluctuations, neck pain, cognitive fatigue), and objective modalities (electroencephalography, electromyography, eye-tracking). Additional citations were manually verified from relevant studies.

Eligibility and Data Extraction

The review included research studies that presented original data from randomized trials, controlled interventions, observational cohorts, and laboratory experiments in one or more mechanistic areas related to digital eye strain. The review excluded narrative commentaries, single-case reports, conference abstracts without full papers, and studies lacking relevant physiological or clinical outcome measures.

The review extracted data on study design, population characteristics (age, setting), domain, exposure details (device type, task duration), and principal outcome measures from each included article. For clinical relevance, studies were grouped into six a priori mechanistic domains: ocular surface and blink dynamics; accommodation and vergence dysfunction; musculoskeletal and ergonomic factors; device-related optical stress; cognitive load; and neurophysiology.

Quality Assessment

The Mixed Methods Appraisal Tool (MMAT) was used to assess the methodological quality of the included studies (Supplementary Table S1). Table 1 illustrates the distribution of study designs across the literature. The MMAT was selected to allow the concomitant appraisal of the qualitative, quantitative, randomized, and nonrandomized, and mixed-methods studies included in this review.[24] Strict adherence to the MMAT operational manual minimized subjective bias and ensured consistency during the quality appraisal conducted by a single reviewer. Rather than excluding studies based on a rigid cutoff score, the MMAT framework systematically identifies and documents specific limitations (eg small sample sizes, short exposure durations (<60 minutes), lack of objective biomarkers, or reliance on self-reported symptoms). The review narratively synthesized these limitations to determine the certainty of evidence within each mechanistic domain.

Data Synthesis

PRISMA flowchart details the study selection process. (Figure 1). Furthermore, the final review included 128 studies (Supplementary Table S2). The synthesis was narrative and mechanism-centered. The findings are summarized in each domain to highlight convergent evidence across methodologies (such as linking laboratory blink data with epidemiological dry eye findings) and mapped to practical interventions. A quantitative pooling (meta-analysis) was not performed due to substantial heterogeneity in the study populations, task paradigms, and exposure durations.

Table 1 Study Type Distribution via Mixed Methods Appraisal Tool

Study Design	Number of Studies
Randomized Controlled trials	45 (35%)
Non – randomised controlled trials	54 (42%)
Quantitative descriptive study	28 (22%)
Qualitative study	1 (1%)

Note: The table illustrates the proportion of study designs evaluated using the Mixed Methods Appraisal Tool.

https://doi.org/10.2147/OPTO.S586750

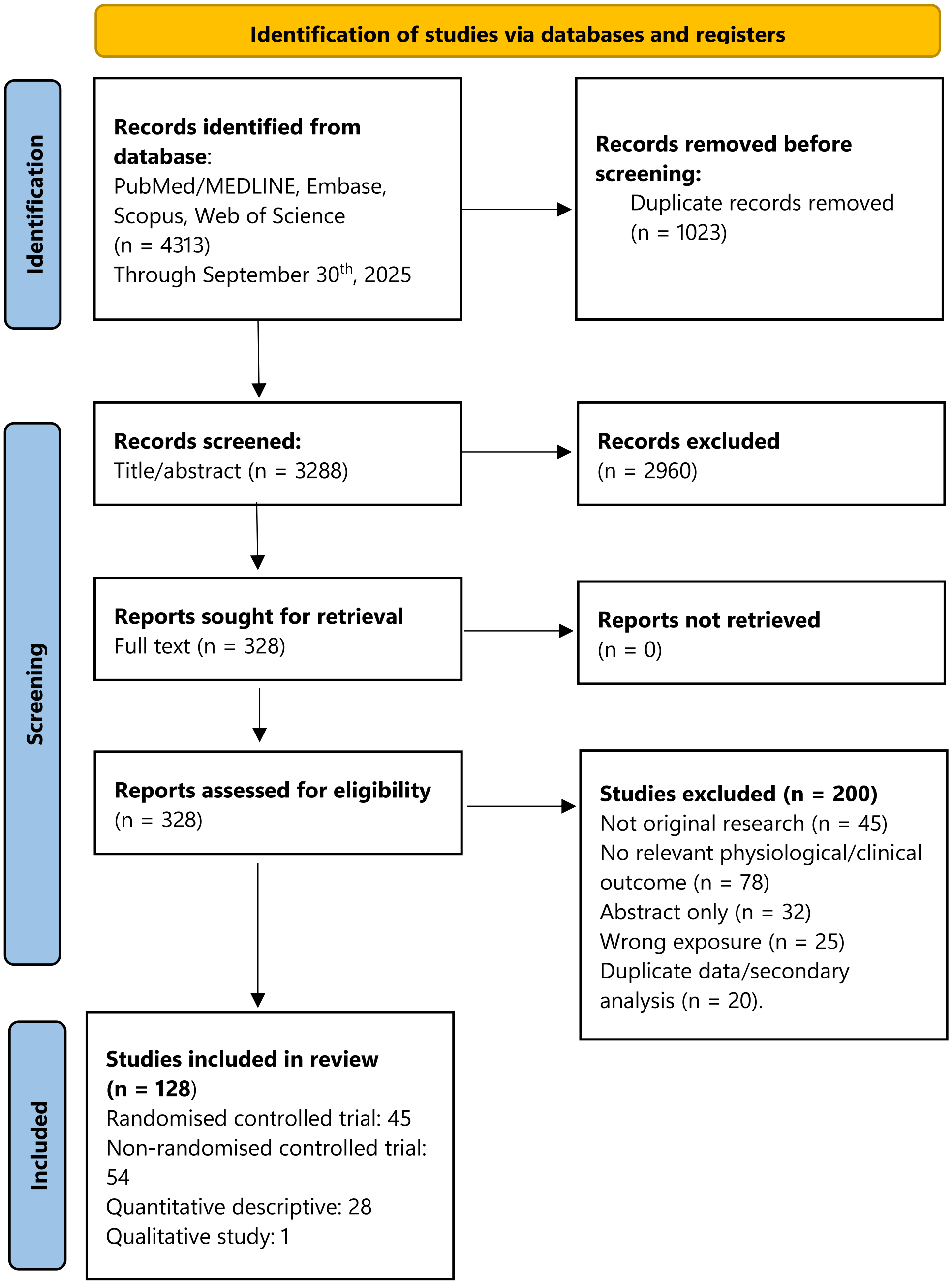


Figure 1 Study selection process. PRISMA flow diagram illustrating the study selection process, including the number of records identified, screened, assessed for eligibility, and included in the final review.

Results

This review included 128 peer-reviewed studies after screening and verification. The final synthesis included 128 studies comprising randomized controlled trials (n = 45), non-randomized controlled trials (n = 54), quantitative descriptive studies (n = 28), and one qualitative study (n = 1). To facilitate clinical interpretation, findings are organized into 6 mechanistic domains: ocular surface and blink dynamics; accommodative and vergence function; musculoskeletal and ergonomic factors; device-related optical stress; cognitive load; and neurophysiological factors. Additionally, several individual risk modifiers (age, refractive error, contact lens wear, gender, psychosocial stress, environment) influencing vulnerability and symptom severity have been identified. Figure 2 summarizes the distribution of included studies across the six mechanistic domains. The results are presented according to study design, key outcomes, and optometric

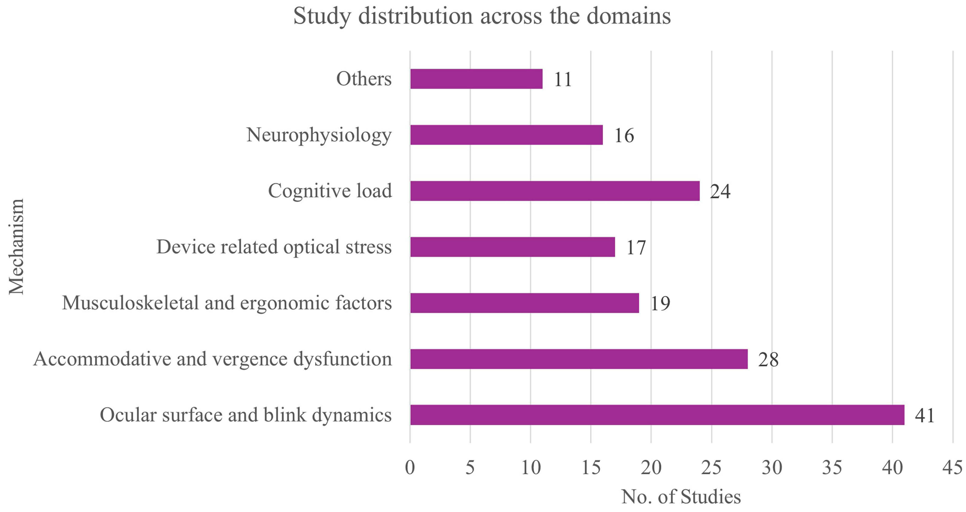


Figure 2 Study distribution across mechanistic domains in digital eye strain. Distribution of included studies across mechanistic domains implicated in digital eye strain. The horizontal bar chart displays the number of studies mapped to six predefined mechanisms. Ocular surface and blink dynamics constituted the largest evidence cluster (41 studies), followed by accommodative and vergence dysfunction (28 studies) and cognitive load (24 studies). Musculoskeletal and ergonomic factors (19 studies), device-related optical stress (17 studies), and neurophysiology (16 studies) showed moderate representation, while the "Others" category (11 studies) includes studies addressing mixed or unclassified mechanisms.

relevance with each domain, with particular attention to age-stratified vulnerabilities and translational implications for clinical practice. This structure enables a mechanism-based synthesis that aligns with the proposed optometric pathway for digital eye strain management.

Ocular Surface and Blink Dynamics

Blinking is a neuromuscular reflex that maintains ocular surface protection, preserves tear film stability, and aids visual fixation. Blinking occurs through brief suppression of the levator palpebrae and activation of the orbicularis oculi, with the eyelid position further supported by Müller's muscle and the tarsal plate. Any disturbance in this meticulously coordinated mechanism, whether due to neuronal anomalies or muscular irregularities, can hinder the blink rate and jeopardize ocular surface stability. Evinger et al further demonstrated that eyelid movements arise from distinct muscular and passive mechanisms, with levator activity driving upward lid excursions and passive forces and orbicularis contraction mediated downward closure. These normative kinematic data provide a valuable reference for the clinical diagnosis of lid motility disorders, reinforcing the importance of blink mechanics in ocular surface health.[25]

Across randomized, experimental, and observational studies, researchers primarily identified reduced blink rate, increased incomplete blinking, shortened tear breakup time, and task-dependent tear film instability as key ocular surface manifestations of digital eye strain. Numerous studies have addressed the role of blinking behavior and tear film stability in digital eye strain. Blink dynamics, including rate, completeness, and tear film stability, are fundamental to optometric assessment, as they determine ocular comfort and visual efficiency during prolonged screen exposure. The evidence is grouped into three mechanistic themes:

Blink Rate and Cognitive Demand

Across multiple studies, the blink rate is strongly modulated by cognitive demand rather than display type: cognitive load reduces the blink rate by approximately 25%–26%, often to 7–10 blinks/min during demanding reading, and refractive error simulations further suppress blinks during video and easy reading conditions; pupil dilation increases in parallel as a workload marker.[2,17,26] Blink-related oscillations (BROs) reliably reflect load differences (including reduced pre-blink activity) and remain robust across rest, visual, and auditory tasks. However, visual stimulation attenuates BRO amplitude versus rest or auditory conditions.[27,28] Task dynamism amplifies these effects: fast-paced visual activities can suppress the blink rate to approximately 33% of baseline and increase incomplete blinks.[8,29] Time course investigations of prolonged VDT use reveal a distinct sequence: early dominance of incomplete blinks, a decrease in the ring breakup time (RBUT) / noninvasive breakup time (NIBUT) declines within 30 minutes, and parallel increases in anterior corneal aberrations.[30] Device type and posture further shape blink suppression: occupational computer use, sustained down gaze, and immersive AR/VR displays produce larger reductions in blink frequency, whereas habitual short viewing distances compound the effect.[31–35] Dry eye patients often exhibit a higher blink frequency, yet a greater proportion of these blinks are incomplete, which disrupts tear film stability and exacerbates symptoms.[36]

Incomplete Blinking and Ocular Surface Damage

Incomplete blinking was consistently associated with ocular surface compromise. Collins et al reported incomplete blinks in both normal eyes and contact lens wearers, with contact lens users showing greater inferior corneal staining and therefore greater susceptibility.[37] Portello et al found that greater symptom severity was associated with a higher proportion of incomplete blinks and with a lower blink rate.[7] Argilés et al reported that electronic reading increased the incomplete blinks (14.5% vs 5% for print),[6] and Abusharha reported that the blink rate decreased for both tablet and paper readings with similar symptom levels.[34] Himebaugh et al demonstrated that reduced and incomplete blinking undermines tear-film replenishment—particularly in dry-eye patients—linking blink mechanics to tear film destabilization.[36] Cardona et al reported that blink regularity, not just rate or completeness, significantly increases ocular surface exposure during reading tasks, underscoring the need to assess blink quality when advising patients on blink reeducation.[29] During 60 minutes of smartphone reading, incomplete blinks progressively increased and correlated with worsening ocular comfort and a reduced binocular accommodative facility.[35] Beyond tear film disruption, blinks may also transiently suppress visual sensitivity. Ridder and Tomlinson demonstrated that contrast sensitivity is reduced immediately after a blink, particularly at low spatial frequencies, with recovery following a time course similar to saccadic suppression, supporting the hypothesis of a shared neural mechanism and highlighting the perceptual consequences of blink timing during sustained digital tasks.[38]

Dry Eye and Tear Film Stability

Tear film instability is a central mechanism that connects sustained digital device use to both ocular discomfort and functional visual decline. Clinical and experimental studies have shown that digital tasks and symptomatic dry eye reduce tear film breakup times, impair lipid layer function (often via meibomian gland dysfunction),[39,40] and produce measurable visual consequences such as reduced contrast sensitivity and increased higher-order aberrations.[31,41–43] Kaido et al demonstrated that ocular surface damage in aqueous-deficient dry eye triggers stress responses detectable by EEG and that short BUT dry eye provokes abnormal accommodative microfluctuations and visual fatigue.[44] Cross-sectional work in computer users reported high symptom prevalence and strong associations between daily screen hours and ocular surface disease index (OSDI) scores,[41] with noninvasive breakup metrics: tear breakup time (TBUT) / post blink blur time (PBBT) markedly lower in symptomatic groups, supporting their routine use in screening.[42,43]

Altered blink behavior, increased incomplete blinks, and irregular blink patterns — prolonged ocular surface exposure and accelerated tear film, resulting in localized lipid layer disruption and faster aqueous evaporation.[29,31–36]

Time-course and task-based experiments show rapid deterioration of tear film metrics during sustained near work: RBUT/NIBUT often falls within the first 30 minutes of VDT exposure, and parallels rises in anterior corneal higher-order aberrations.[30] Smartphone and prolonged reading paradigms produce similar patterns over 60 minutes, with progressive increases in incomplete blinks and reduced tear stability that correlate with worsening comfort and reduced binocular accommodative facility.[35] Task-based work has also shown that post-task NIBUT reductions impair binocular fusion maintenance.[45] Mechanistic studies have revealed that rapid inflammatory responses to desiccating stress (mitogen activated protein kinase (MAPK), cytokines, and matrix metalloproteinase −9 (MMP-9)) are involved in these changes.[46] Additionally, Ayaki et al reported a 31.8% prevalence of eye strain among a large cohort. They identified short TBUT, superficial punctate keratitis, and a thinner ganglion cell complex as significant risk factors.[47]

Accommodative and Vergence Functions

Multiple randomized, experimental, and observational studies have characterized the involvement of accommodation and vergence in digital eye strain primarily through increased accommodative lag, reduced accommodative and vergence facility, reduced fusional reserves, and increased accommodative microfluctuations during prolonged near work. These changes in function indicate the ongoing accommodative and vergence requirements resulting from prolonged near digital tasks, especially in visually underdeveloped or binocularly susceptible groups. Prolonged near work creates ongoing demands on accommodative and vergence systems, which can surpass physiological limitations, leading to symptoms and quantifiable decreases in function.[23,48] The evidence is grouped into three mechanistic themes:

Accommodative Lag and Fatigue

Consistent near tasks using smartphones and other small displays increase accommodative lag, reduce amplitude and facility, and induce transient accommodative fatigue, with the effects being most potent in adolescents and young adults.[9,10,15,49] Ethoven et al used objective smartphone tracking in Dutch teenagers and found that frequent continuous use episodes (≥20 minutes) were significantly associated with more myopic spherical equivalent refraction and higher axial length-to-corneal radius ratios, reinforcing the role of sustained near exposure in adolescent refractive vulnerability.[50] Padavettan et al reported that short-term smartphone reading produced a measurable increase in accommodative lag and reduced relative accommodation,[9] whereas Park et al reported that prolonged smartphone exposure caused transient accommodative fatigue in young adults;[49] Chellapan et al reported a reduced amplitude, facility, and positive relative accommodation in teenagers,[15] and Kang et al reported that smaller displays exacerbate near point deterioration compared with larger devices.[15] Device and display characteristics (size, spatial frequency, and contrast) modulate strain, and population factors alter the prevalence and phenotype: Berens and Stark noted objective and subjective variability across occupational groups,[51] and Reindel et al reported high rates of ill-sustained accommodation and accommodative insufficiency among symptomatic myopic contact lens wearers, indicating that mid life lens users may show distinct vulnerability.[52]

Vergence Anomalies and Binocular Dysfunction

Smartphone use and other near-device tasks can impair vergence function. However, the effects vary by age, task duration, and baseline binocular status. Padavettan et al reported that short-term smartphone reading reduced fusional vergence and vergence facility,[9] and Chellapan et al reported deterioration in the near point of convergence and fusional reserves in adolescents,[10] highlighting pediatric vulnerability. In contrast, Allen and Mehta reported no significant change in accommodative or convergence measures after 30 minutes of smartphone use,[53] highlighting the variability in samples and protocols. Collier et al reported that small fixation disparities are associated with greater discomfort during computer work, suggesting that even subtle binocular misalignment modulates symptoms.[54] For clinical screening in community settings, a minimal battery of near point of convergence, monocular accommodative facility, and phoria differences is practical and sensitive to non-strabismic binocular vision anomalies.[52,55]

Accommodation–Vergence Incongruence and Cross-System Load

Zetterberg et al reported that a mismatch between accommodation and convergence, rather than accommodation alone, significantly increases trapezius muscle activity during visually demanding near work, implicating binocular incongruence as a driver of neck and shoulder load.[56] Subsequent work demonstrated that high cognitive demand at short viewing distances amplifies this effect by increasing accommodative lag and blink suppression alongside trapezius activation, reinforcing a visual–postural coupling mechanism.[20] Ergonomic and physiological studies have extended this pathway: Domkin et al reported that very near viewing elevates both ciliary muscle contraction and trapezius activity, supporting a ciliary-to-postural link,[57] and Richter et al reported progressive trapezius activation during sustained near tasks, suggesting that sustained visual attention is an additional musculoskeletal driver.[58] Practical ergonomic thresholds (for example, head flexion of > 20°) further magnify visual–postural coupling and discomfort.[59] These findings support the assessment of binocular mismatch as part of a broader cross-system evaluation for patients with near-task-related neck and shoulder symptoms.

Accommodative Microfluctuations and Optical Factors

Accommodative microfluctuations are small, continuous oscillations in the accommodative response of the eye around a steady focus; they reflect dynamic ciliary muscle activity and the feedback control loop that maintains focus during sustained near or intermediate viewing.[60] These microfluctuations have emerged as sensitive markers of visual fatigue and cognitive load: myopic juveniles show unstable microfluctuations during sustained near work that peak at approximately 30 minutes,[11] and cognitive demand selectively increases the high-frequency component of microfluctuations, thereby linking them to autonomic arousal.[60] In contrast, some short-term VDT studies report stable microfluctuations and pupil size, suggesting that task duration and display type determine whether microfluctuations index fatigue or reflect display quality.[16] Optical and ergonomic factors further modulate microfluctuations: downward gaze alters ocular aberrations during accommodation,[61] while stimulus design and spatial frequency affect accommodative response,[62] chromatic cues drive accommodation under narrowband lighting,[63] and higher-order aberrations can reduce the directional signal from microfluctuations, constraining reflex accommodation and increasing inter-individual variability.[64] Display format and temporal coding also influence high-frequency responses and subjective fatigue, with 3D and time-sharing displays producing larger microfluctuation responses than 2D displays do.[65] Lens optics also matter: aspheric contact lens designs produce more minor microfluctuation changes than spherical designs do, suggesting a potential optical mitigation strategy.[66] Finally, ocular surface instability is directly linked to elevated microfluctuations: short BUT dry eye is associated with higher blink frequency and increased high-frequency microfluctuation components, providing a mechanistic bridge from tear film disruption to unstable accommodation and symptom generation.[67,68]

Musculoskeletal and Ergonomic Factors

Musculoskeletal symptoms of the neck and shoulders are a common and functional key component of digital eye strain. Epidemiological surveys have reported a high prevalence of neck/shoulder pain in students and workers, and device type, prolonged use, close viewing distance, prior pain history, and psychosocial stress have been identified as consistent risk factors.[12–14] Objective electromyographic and postural studies further demonstrated increased trapezius activation, forward head posture, and elevated neck–shoulder load during visually demanding digital tasks, particularly under conditions of binocular incongruence and short viewing distance. Objective behavior matters, as symptomatic users adopt closer viewing distances (approximately 30 cm for phones and 56 cm for computers) than asymptomatic users, supporting minimum-distance guidance in clinical counseling.[69]

Controlled laboratory studies clarify visual–postural coupling. Zetterberg et al showed that incongruence between accommodation and convergence, rather than accommodation alone, significantly increased trapezius electromyography (EMG), implicating binocular mismatch as a driver of neck/shoulder activation.[56] Domkin et al reported that very near viewing (25 cm) stimulated greater ciliary muscle contraction and increased trapezius activation, consistent with a mechanistic link between ciliary effort and postural load.[57] Richter et al observed that the activity of the trapezius muscle increases over time during sustained near tasks across lens conditions, indicating that prolonged visual attention and cognitive effort also increase the musculoskeletal load.[58,70] Frequent short walking breaks during prolonged sedentary work improved reaction time, mood, and alertness while reducing prefrontal activation, thereby complementing visual micro breaks with movement.[71] Visual stressors, such as glare, induce orbicularis and trapezius responses, thereby linking visual discomfort to sympathetic/muscle activation.[72] Lin et al found that severe discomfort glare produced faster eye movements and greater pupil constriction, with subjective glare ratings strongly predicting eye movement speed and moderately predicting pupillary constriction.[73] The direction of gaze and optical changes also influence visual load: looking downward while focusing up close can gradually change higher-order aberrations over several minutes. This factor should be considered when evaluating ergonomics for tasks performed at close range.[61] According to Abu-Ghosh et al forward head posture significantly increases dual-task cost across gait metrics, indicating higher cognitive load during concurrent motor tasks. This cognitive–motor coupling supports posture correction, thereby reducing both musculoskeletal and executive burdens.[57,74]

The workstation configuration and posture substantially influence the objective load. Dahlqvist et al demonstrated that a single 49-inch curved screen positioned at 90 cm resulted in the lowest trapezius load and the most neutral head posture compared with standard multiscreen setups.[75] Chen and Chan demonstrated that head flexion beyond 20° and unsupported sitting markedly increase neck-shoulder muscle activity and discomfort, providing simple clinical posture thresholds.[59] Postural misalignment affects shoulder mechanics even in asymptomatic individuals. Thigpen et al reported that forward head and rounded shoulder posture (FHRSP) significantly altered scapular kinematics and reduced serratus anterior activity during overhead tasks. These biomechanical changes support the FHRSP's role as an intrinsic risk factor for shoulder dysfunction, reinforcing the need for postural screening in digital eye strain management.[76] Environmental ergonomics also play a role: higher ambient temperatures impair cognitive performance, reduce parasympathetic activity, and increase subjective workload even within thermally comfortable ranges, with cooling strategies such as fans or ventilation mitigating these effects.[77] Young adults in non-air-conditioned buildings show cognitive decline during heat waves, underscoring the importance of indoor temperature regulation for sustaining performance under extreme environmental stress.[78]

Device-Related Optical Stress

The primary characteristics of digital eye strain associated with device use included increased visual fatigue, alterations to accommodative stability, reduced contrast sensitivity, and heightened discomfort related to display luminance, flicker, spectral composition, and viewing geometry, as found in both experimental and observational studies. Display dynamics, presentation methods, and habitual use patterns increase accommodative-vergence demand, destabilize the tear film, and amplify cognitive and musculoskeletal loads, thereby causing device-related optical stress. Empirical data show that smaller displays and shorter viewing distances accelerate accommodative/convergence strain and earlier symptom onset.[1,9,15,69] In contrast, larger single curved monitors positioned at ergonomic distances reduce the trapezius load and produce a more upright head posture than multiscreen setups.[56,57,70,75] Temporal presentation and flickers remain important drivers of perceptual and cortical strain. High spatial-frequency edges and natural eye movements can render flickers perceptible well above conventional fusion thresholds, supporting the use of high native refresh rates or pulse-width modulation (PWM) alternatives for symptomatic users.[79,80] Yoshimoto et al used paired comparisons of flickering stimuli and found that visual discomfort increased with deviations from natural 1/f amplitude spectra and was significantly higher for random-phase flicker than for fixed-phase flicker.[81] Optimizing display luminance to ambient illuminance minimized visual fatigue while preserving performance and comfort, supporting adaptive dimming that tracks room light.[82] Recent ergonomic modeling reinforces this, establishing that specific display luminance levels and contrast polarity (light vs dark mode) must be tuned to the surrounding office lighting to achieve the lowest visual fatigue scores.[83] In practice, this indicates that patients may perceive flicker or blur when displays do not mimic natural visual patterns.

Spectral content and luminance relationship modulate both immediate performance and circadian physiology—acute spectral tuning that reduces short-wavelength retinal illuminance lowers EEG fatigue markers and dry-eye indicators,[84,85] and recent experimental work has shown that short-wavelength-rich light-emitting diode (LED) exposure can impair contrast sensitivity at mid-to-high spatial frequencies with partial preservation of thresholds by blue blocking lenses.[86] Beyond acute performance, high-energy visible blue light (450–480 nm) has been implicated in delayed post-task neural recovery.[87] This phenomenon, characterized by a sluggish alpha power rebound after screen exposure ceases, suggests that blue-enriched spectra may cause "incomplete cortical restitution", prolonging the physiological experience of fatigue even during rest.[88]

However, this benefit appears to be limited to visual performance. A recent randomized crossover study found no measurable benefit of blue-filtering lenses for dry eye signs or symptoms during a 120-minute reading task,[89] suggesting that spectral filtering is unlikely to be an effective standalone therapy for ocular surface disease in short-term exposures.

Immersive modalities (3D/VR/AR) amplify oculomotor suppression and modality-specific EEG signatures of fatigue. Kim et al reported that blink rates dropped significantly lower in VR headsets (approximately 10.8 blinks/min) than in natural views (18.0 blinks/min), indicating a heightened risk of desiccating stress.[33] These modalities require graded exposure, reduced stereoscopic disparity, and objective monitoring when used for prolonged sessions.[90–92] The neurophysiological impact of digital strain appears to be topography-specific depending on the immersion level. Lee et al mapped these distinct signatures, revealing that 2D and 3D displays primarily elevate prefrontal delta–theta activity (Fp1, Fp2), whereas AR modulates occipital alpha rhythms (O1, O2), and VR triggers central delta–theta elevation (C3).[91]

Recent measurements show that organic-light emitting diode (OLED) smartphone displays using 240 Hz PWM may flicker at very close distances. However, this effect quickly fades beyond a few centimetres, indicating minimal risk for typical use. However, caution is advised for sensitive individuals or unusually close viewing.[93]

Cognitive Load and Neurophysiology Factors

Experimental studies manipulating task complexity showed that increased cognitive load amplified blink suppression, accommodative instability, and subjective fatigue, independent of display type. Cognitive demand and neurophysiological state were consistently associated with concurrent modulation of subjective workload, peripheral ocular physiology, and cortical activity, supporting their role as cross-system contributors to digital eye strain (Table 2). Experimental studies consistently show that blink rate decreases with increasing cognitive load, independent of display type, indicating that mental effort rather than screen format drives ocular suppression.[2] When visual stressors, such as refractive error or low contrast, combine with high cognitive demand, internal and external asthenopic symptoms increase, and the blink rate falls further, demonstrating a synergistic interaction between optical degradation and mental effort.[17] Beeson et al documented progressive worsening of visual-ocular symptoms and reduced productivity during 60 minutes of digital tasks under higher cognitive load, while task accuracy remained stable This time-course pattern supports the use of scheduled micro-breaks and workload modulation before errors emerge.[1] Compared with a single timed break, frequent or self-paced micro-breaks during 40-minute laptop reading reduce eyestrain and stabilize accommodative variability, reinforcing break cadence over fixed timing as a practical management variable.[94]

Table 2 Cognitive Load and Neurophysiology Mechanisms in Digital Eye Strain

Mechanism	**Key Findings**	**Study Designs**	**Clinical Implications**	**Recommended Interventions**
Frontal Theta Activity	Increased frontal theta during demanding digital tasks; early marker of cognitive fatigue.[95–97]	EEG-based lab studies	Early, objective indication of central fatigue before symptoms emerges	Task pacing, warm light, reduced multitasking
Occipital Alpha Suppression	Reduced occipital alpha power during visually demanding tasks and flicker exposure; linked to sustained attention and visual strain[44,88,98]	EEG studies, cognitive load paradigms	Indexes visual effort; modulated by display properties and spectral content	Spectral tuning, flicker optimization, structured breaks
Accommodative Micro fluctuations	High-frequency micro fluctuations increase with cognitive demand and visual fatigue; unstable in myopic juveniles during sustained near work[11,60,67,68]	Eye-tracking and cognitive load studies	Indicates visual fatigue and unstable accommodation	Aspheric/optimized optics, reduced continuous near duration, accommodative training
Blink Suppression Under Cognitive Load	Blink rate decreases and incomplete blinks increase with higher cognitive demand[2,6,8,17]	VDT experiments, reading tasks	Contributes to tear film instability and ocular surface exposure	Blink training, lubricants, task structuring to avoid prolonged high-load intervals
EEG–Ocular Surface Coupling	Dry eye and short BUT provoke frontal EEG stress responses; associated with visual fatigue and accommodative instability.[44,45,68]	EEG and ocular surface studies	Supports neuro-ocular feedback loop	Integrated dry-eye management plus workload/lighting adjustments
VR/AR-Specific EEG Fatigue Signatures	Immersive VR/AR displays show stronger fatigue markers, altered regional brain activity, and reduced blink rate[32,33,90,91,99]	Immersive device experiments with EEG and eye-tracking	Higher risk of desiccating stress and central fatigue	Graded exposure, reduced stereoscopic disparity, session time limits, monitoring in vulnerable users

Notes: Cognitive and neurophysiological mechanisms contributing to digital eye strain. The table synthesizes evidence from EEG, accommodative, and blink-behavior studies, highlighting mechanistic pathways, clinical relevance, and intervention strategies supported by the included literature.
Abbreviations: EEG, electroencephalography; BUT, breakup time; VR, virtual reality; AR, augmented reality; VDT, visual display terminal.

Cognitive demand also influences the accommodative control. Hynes et al reported that the high-frequency component of accommodative microfluctuations increases systematically with task difficulty, suggesting that cognitive load augments ciliary muscle activity and autonomic arousal during near work.[60] Mihelčič and Podlesek showed the distribution of load across task-type shapes: stroop tasks produced the largest pupil diameters, consistent with conflict-related arousal, whereas textual logical reasoning tasks induced the most significant accommodative fluctuations, indicative of near-point stress.[100] Developmental and refractive influences further modulate these responses. Roberts et al reported that cognitive engagement boosts accommodative accuracy and reduces variability in children. Conversely, leaving hyperopia uncorrected destabilizes sustained near tasks, showing that both the material presented and the correction status shape how cognitive load manifests in the accommodative system.[101] Together, these findings position accommodative microfluctuations and pupil dynamics as sensitive ocular markers of mental workload, complementing blink-based indices.[60,100,102]

Neurophysiological Factors

Neurophysiological studies using EEG, ERP, and VEP paradigms identified objective markers of central fatigue, including latency prolongation, amplitude attenuation, and altered cortical oscillatory activity during sustained digital tasks. EEG and BROs provide objective neural markers of workload and fatigue that often precede subjective awareness. Researchers observed task-related desynchronization of posterior alpha rhythms (8–12 Hz) and relative increases in theta (4–7 Hz) and delta (1–3 Hz) activity during prolonged or cognitively demanding digital tasks, indicating heightened cortical effort and reduced processing efficiency.[103] Under increasing cognitive load, multitasking paradigms show characteristic changes in frontal theta and alpha power, with lower power in these bands associated with better performance, indicating that simple power measures must be interpreted alongside behavior.[95] Gamma-band features extracted from short EEG segments can reliably distinguish single-task from multitask states, highlighting their value for classifying high-load conditions.[104] Blink-related oscillations add a naturalistic window into workload: Page et al demonstrated that BRO responses differentiate low, medium, and high workload in a multi-attribute task battery, including reduced pre-blink theta desynchronization at higher load,[27] while Liu et al showed that BRO topography differs between visual and auditory environments, with occipital–parietal dominance during visual tasks and frontal–temporal dominance during auditory tasks.[28,99] These findings support BROs as discreet markers of modality-specific cognitive engagement in everyday tasks.

Across electrophysiological modalities, convergent markers of central fatigue and workload emerged despite heterogeneity in task paradigms and exposure durations. ERP studies consistently demonstrated prolongation of cognitive processing latencies—most prominently involving N200 and P300 components—alongside attenuation of P300 amplitudes during sustained attention, high cognitive load, or prolonged digital task engagement, reflecting delayed stimulus evaluation and reduced attentional resource allocation.[95,96,102–104] VEP investigations similarly reported prolongation of early cortical response latencies, particularly P100, with concurrent reductions in N75–P100 amplitudes during visually demanding or prolonged near-viewing conditions, indicating slowed visual cortical processing efficiency under sustained digital exposure.[84,87,91] EEG studies further revealed task-dependent reductions in posterior alpha power accompanied by relative increases in frontal theta and low-frequency activity during extended or cognitively demanding digital tasks, patterns repeatedly associated with mental fatigue, sustained attentional demand, and reduced processing efficiency.[88,97,105–109] Notably, several studies reported that these electrophysiological changes preceded subjective symptom awareness, supporting EEG- and ERP-based measures as objective, early markers of neural fatigue in digital eye strain.[27,28,99,110]

Event-related and network-level EEG metrics further refine the understanding of visual and cognitive stress in digital environments. Gulbinaite et al showed that a 10 Hz flicker entrains alpha oscillations and impairs selective attention, particularly on incongruent trials requiring high selective control, implying that exogenous alpha-band stimulation can disrupt endogenous attention when the frequencies align.[88] Zhang et al reported that 40 Hz visual flicker increases occipital gamma power and alters EEG microstate complexity, paralleling patterns seen in neurological disease and suggesting that high-frequency visual stimulation can systematically reshape cortical dynamics.[98] In a large auditory oddball cohort, Studenova et al demonstrated that P300 amplitude and latency are tightly linked to stimulus-triggered alpha attenuation, supporting the view that classic evoked responses reflect modulation of non-zero-mean ongoing alpha activity rather than independent components.[105] Mixed-reality and immersive displays add a network perspective: Wu Yan et al found that stereoscopic depth motion in mixed-reality increased phase-locked values and strengthened brain connectivity across alpha, theta, and delta bands, with higher betweenness centrality and nodal efficiency in frontal, temporal, parietal, and occipital regions, validating EEG-derived network topology as a marker of visual fatigue in mixed-reality environments.[99] Lee et al showed that screen size and presentation mode differentially affect regional EEG activity: 2D/3D stimuli mainly altered prefrontal delta–theta power, augmented reality modulated occipital alpha, and virtual reality elevated central delta–theta activity, with larger screens and VR inducing the greatest visual fatigue and, often, motion sickness.[91] Collectively, these findings underline that both temporal (flicker, frequency) and spatial (field, immersion) properties of digital displays can entrain or disrupt endogenous oscillatory dynamics relevant to attention and fatigue.[88,91,98,99]

Mobile EEG and related modalities demonstrate that cognitive fatigue can be tracked in real time during realistic high-demand tasks, frequently outperforming self-report. Wylie et al used signal detection theory during repeated n-back tasks to show that as fatigue increased, participants adopted more conservative response criteria and exhibited reduced perceptual certainty, with striatal activation correlating with both fatigue and behavioral measures.[106] Wascher et al and others have shown that mobile EEG is feasible in actual workplaces, capturing fatigue-related changes without disrupting tasks.[97] Krigolson et al found that mobile EEG biomarkers of cognitive fatigue in medical students during a simulated night-on-call shifted markedly from the start to the end of the shift, despite poor correspondence with self-reported fatigue scores; this “fatigue blindness” underscores the limitations of symptom-based management and the potential safety benefits of objective monitoring.[96] Complementary modalities refine this picture. Gani et al reported that removing corrective lenses in myopic participants elevated frontal midline theta and suppressed occipital alpha during visual stimulation, indicating that retinal blur forces the cortex into a higher-effort compensatory state.[110] Reilly et al showed that task-evoked pupillary response functions are linear and largely independent of baseline pupil size, supporting the use of subtractive baseline correction and confirming pupillometry as a reliable autonomic correlate of cognitive effort, which can be meaningfully combined with EEG markers.[102] Neurofeedback work suggests some degree of short-term trainability: Quaedflieg et al demonstrated modulation of frontal alpha asymmetry during training in a frequency- and location-specific manner. However, effects were variable across individuals and did not persist at the one-week and one-month follow-ups, suggesting limited long-term stability.[107]

Environmental, circadian, and musculoskeletal factors further interact with cognitive state to shape digital eye strain and its neural correlates. Higher ambient temperatures within nominally comfortable ranges (24–28 °C) have been shown to reduce cognitive performance, decrease parasympathetic activity, and increase subjective workload. At the same time, young adults living in non-air-conditioned buildings during heat waves exhibit slower reaction times and reduced throughput on cognitive tasks compared with peers in air-conditioned housing.[77,78] Evening exposure to LED-backlit screens suppresses melatonin. It reduces sleepiness while acutely enhancing attention and memory, raising concerns about circadian misalignment with chronic use.[111] Spectral tuning offers partial mitigation: blue-shifted smartphone displays adjusted toward longer wavelengths reduce retinal photoreceptor illuminance for S-, M-, L-cones and ipRGCs, and are associated with lower dry-eye symptom scores and EEG fatigue markers than standard LCD or OLED conditions.[84] Chronotype modulates these effects, with afternoon blue light eliciting distinct theta and alpha1 responses in morning-type individuals, underscoring the need for personalized lighting guidance.[87] Beyond screens, intermittent bright light pulses can modestly improve cognitive performance and mood but are often perceived as less comfortable In contrast, multi-colour light-filtering glasses reduce frontal beta power, suggesting a downshift in cortical arousal and potential relaxation benefits in visually demanding environments.[108,109]

Posture and movement contribute additional load channels that converge with visual and cognitive strain. Forward head posture significantly increases dual-task cost during walking, indicating impaired sensorimotor integration and higher executive demand even in ambulatory tasks.[74] Laboratory work shows that trapezius muscle activity rises during sustained near tasks regardless of accommodative and vergence demand level, supporting a model in which visual attention and cognitive effort, rather than optics alone, drive neck–shoulder activation.[70] Taken together, this body of evidence positions cognitive load as a central amplifier of digital eye strain, where mental effort, environmental context, posture, and cortical dynamics jointly determine how aggressively digital tasks drive ocular discomfort, musculoskeletal strain, and performance decline.

Individual Risk Modifiers

Uncorrected refractive error and myopia progression are major amplifiers of digital eye strain at the individual level. Prolonged near and smart device use is associated with increased axial length and myopic spherical equivalent. Enthoven et al showed that continuous smartphone use episodes were significantly associated with greater myopic refractive error and a higher axial length-to-corneal radius ratio, particularly among teenagers with low outdoor exposure, underscoring the importance of breaks and outdoor activity in myopia prevention.[50] A targeted school intervention reduced eyestrain symptoms. It slightly slowed myopic progression in children with refractive errors, aligning pediatric visual hygiene with refractive stability.[112] Longitudinal evidence indicates that longer daily smartphone use accelerates myopia progression. In contrast, greater outdoor time and increased viewing distance are protective.[113–115] Short-term nearwork can induce myopic shifts and anterior segment changes, which are preventable with virtual distant-view displays, highlighting the causal plausibility and ergonomic mitigation.[116]

Optical correction strongly modulates symptom burden and neural processing: appropriate refractive correction reduces subjective fatigue, improves vision-related quality of life, and normalizes cortical recruitment during complex visual tasks, whereas uncorrected error increases cortical effort and exacerbates difficulties in vision-related tasks.[110,117–119]

Presbyopia and age-related decline increase near-task effort and symptom prevalence among middle-aged workers. Computer users with presbyopia report higher CVS scores, with dry eye and a lack of breaks as common ergonomic contributors.[120] Optical aberrations and accommodative instability interact with cognitive demand to amplify perceived effort: short cognitive loads differentially alter pupillary and accommodative responses—conflicting tasks enlarge pupils while logical tasks destabilize accommodation—so higher-order aberrations and repeated microfluctuations increase retinal blur and subjective strain during sustained nearwork.[100]

Systemic health and obesity can affect the ocular surface, and autonomic factors can contribute to fatigue. Higher BMI in children is linked to structural changes in the meibomian glands, shorter tear breakup times, and higher symptom scores, suggesting that systemic inflammation, tear instability, and reduced blink efficacy increase susceptibility to digital eye strain.[121]

Behavioral and developmental factors shape acute symptoms and long-term outcomes: excessive near-bedtime smartphone use worsens sleep quality,[122] early high-screen exposure predicts poorer developmental outcomes,[123] and objective digital behavior metrics (data usage) can reveal associations with refractive status not captured by self-reported time.[115,124,125] Longitudinal data confirm that screen time and reduced sun exposure accelerate myopia progression.[126]

The cognitive load magnifies interactions between visual stressors and symptoms: combining visual stress (refractive error, low contrast) with high cognitive demand increases internal and external asthenopic symptoms and suppresses the blink rate, whereas neural decoding shows that corrective lenses alter subjective fatigue and cortical signatures.[17,117]

Management

Managing digital eye strain is best framed as a menu of evidence-informed options tailored to the likely underlying mechanisms rather than as a prescriptive protocol (Figure 3). A brief targeted assessment linking history (device types, hours viewing distance) with a short test battery (OSDI/DESQ, NIBUT, blink observation, near point of convergence, and accommodation) can identify whether ocular surface instability, binocular dysfunction, or device-related optical stress predominates (Table 3). Clinicians should prioritize objective exposure metrics (eg screen time logs or data usage) over simple self-reports to accurately gauge risk.[114,115]

Ocular Surface and Blink Dysfunction

Conservative measures such as lid hygiene and blink retraining[127] are foundational for the management of ocular surface issues. A single in-office thermal pulsation treatment (TearCare) has been shown to improve reading speed and OSDI scores in patients with meibomian gland dysfunction. However, evidence is currently limited to short-term follow-up.[40]

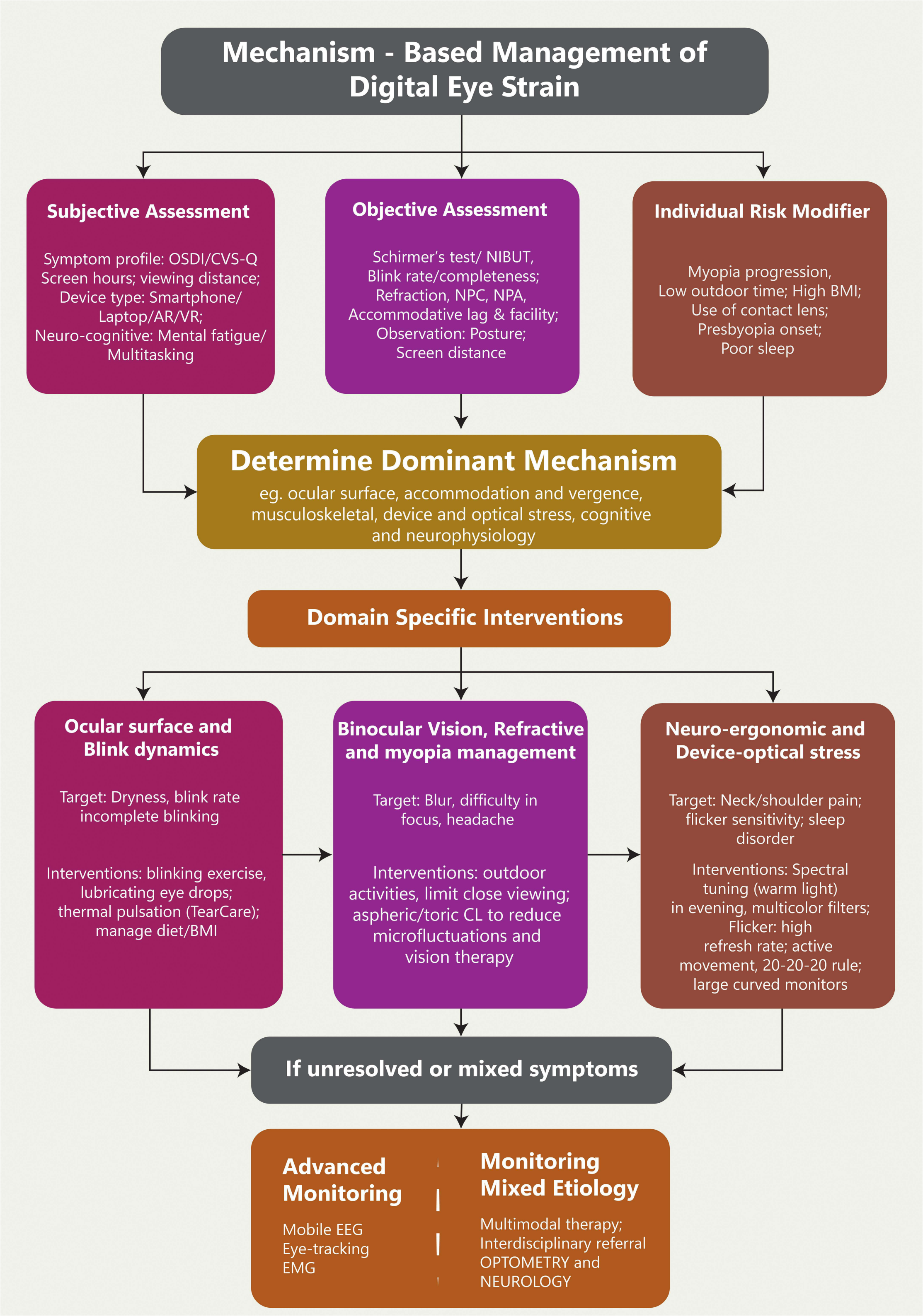


Figure 3 Mechanism-based management of digital eye strain. This flowchart presents a structured approach to digital eye strain by mapping subjective symptoms, objective findings, and individual risk modifiers to dominant mechanisms—such as ocular surface dysfunction, binocular vision stress, and neuro-ergonomic strain. It outlines targeted interventions per domain and recommends advanced monitoring or interdisciplinary referral for unresolved or mixed presentations.

Table 3 Mechanism -Based Management Strategies for Digital Eye Strain

Mechanism Domain	Underlying Driver	Evidence – Based Intervention
Ocular surface	Reduced blink rate and incomplete blinking due to cognitive load	Lid hygiene and blink retraining[127] Thermal pulsation[39] (eg TearCare)[40] Preservative-free lubricants[128]
Optical and binocular	Accommodative lag, microfluctuations, and vergence incongruence	Refractive: Toric or aspheric contact lenses[66,129] Presbyopia: Low-power adds (+0.75 D)[130] Binocular: Office-based vision therapy[131]
Neurophysiology	Cortical fatigue (Alpha/Theta changes) and Cognitive load	Monitoring: Mobile EEG (mEEG) to detect objective fatigue onset.[97] Biofeedback: Use blink-related oscillations (BROs) to trigger breaks.[26] Mindfulness: Brief meditation to enhance cognitive resilience.[132]
Device & Filters	Device-related optical stress (Blue light, Flicker)	Spectral Tuning: Prioritize warm light/spectral tuning for evening use (circadian protection).[111] Blue Light filters/multicolor filters.[86,109] (Standard blue-blocking lenses may not improve dry eye symptoms). Flicker: Use high refresh rates/DC dimming for sensitive users.[79]
Ergonomics and posture	Visual-postural coupling (ciliary-trapezius link)	Large curved monitors (eg 49-inch) to reduce neck load.[75] Maintain viewing distance >30 cm (using biofeedback)[133]
Behavioral Strategy	Static posture and sustained attention	Active Breaks: Prioritize movement (walking/stretching) over passive viewing[71,116,134] Note: Passive 20–20-20 breaks may be insufficient alone[135]
Environment	Ambient factors and circadian disruption	Humidity control (40–60%)[77] Day: Optimize ambient light (300–500 lux)[133] Night: Spectral tuning (reduce blue light) for sleep quality[122]

Notes: The table summarizes evidence-based clinical interventions mapped to the dominant mechanistic drivers of digital eye strain. Each domain—ocular surface, optical/binocular, neurophysiological, device-related optical stress, ergonomics, behavioral strategies, and environmental factors—is paired with its underlying physiological or cognitive driver and corresponding targeted management options. The framework supports mechanism-first clinical decision-making by aligning specific interventions (eg blink retraining, accommodative/vergence correction, spectral tuning, ergonomic optimization, active breaks, and environmental modulation) with the root cause identified during assessment.

Similarly, warming masks have demonstrated objective improvements in lipid layer thickness and complete blink rates in patients undergoing post-refractive surgery.[39] For high-frequency gamers, the prophylactic use of preservative-free hyaluronic acid lubricants combined with breaks has been shown to mitigate symptom onset.[128] A recent comparative study found that although several types of artificial tears—such as carboxymethylcellulose, polyethylene glycol, and hyaluronic acid—can help stabilize the tear film in the short term, drops containing sodium hyaluronate provide better relief from symptoms than those with carboxymethylcellulose.[136] Systemic omega-3 supplementation is effective primarily in screen users with a low baseline omega-3 index.[137]

Optical and Binocular Factors

The correction of a refractive error is critical. Compared with spherical daily disposable contact lenses, toric daily disposable contact lenses have been shown to reduce objective orbicularis oculi muscle activity in patients with low-to-moderate astigmatism.[129] Furthermore, aspheric contact lens designs have been found to reduce accommodative microfluctuations more effectively than spherical designs do, potentially reducing ciliary muscle stress.[66] For presbyopes, lens choice is task-dependent. In contrast, low-power additions (+0.75 D) improve reading speed in general users.[130] Specific "visible display unit (VDU)-progressive" lenses are rated more suitable for computer work than standard progressives in occupational trials.[138] When accommodative insufficiency is identified, office-based vision therapy—potentially augmented with home training devices—yields more stable long-term outcomes than does correction alone.[131]

Physical and Behavioral Interventions

Ergonomic interventions should be extended beyond static postures. While the 20–20-20 rule is standard, active physical breaks appear to be superior to passive rest.[135] Brief static stretching (3 minutes) has been shown to alleviate cognitive fatigue and improve executive function.[139] At the same time, frequent short walking breaks reduce prefrontal cortex activation and preserve working memory better than social breaks.[71] Specific eye-movement tools also show promise; a recent trial of a guided "eye roll" tracking device demonstrated reduced vision discomfort after 4 weeks of use.[134] Furthermore, regarding viewing distance, interventions that utilize larger, curved monitors (eg 49-inch) reduce trapezius load more effectively than dual-monitor setups.[75]

Neurophysiological Monitoring

Emerging technologies offer a "neuroergonomic" approach to management. Mobile EEG (mEEG) has been validated as a reliable, real-time biomarker of cognitive fatigue in workplace and medical simulation settings, often detecting fatigue onset before participants subjectively report it.[96,97] This suggests that management strategies could evolve from fixed schedules (eg every 20 minutes) to objective load monitoring for high-demand users, where breaks trigger physiological markers such as blink-related oscillations or alpha-band attenuation.[27] Beyond monitoring, interventions that incorporate movement and body awareness provide measurable neurophysiological benefits: Heiland et al observed that brief, frequent walking breaks during prolonged sitting reduced right prefrontal oxygenated haemoglobin during demanding working-memory tasks while improving reaction times, mood, and alertness compared with seated social interaction,[71] and Fukuie et al showed that short bouts of static stretching after VDT work increased vitality, arousal, and executive performance relative to rest.[139] At the cognitive level, even brief mindfulness meditation training has been shown to enhance visuospatial processing, working memory, and executive functioning, suggesting a low-cost route to increasing cognitive resilience under sustained digital exposure.[132]

Systemic and Lifestyle Factors

Management must extend beyond the eye to address the systemic contributors to fatigue. Screening for factors such as high BMI is relevant in pediatric populations, as obesity has been linked to meibomian gland structural changes and tear instability.[121] Furthermore, clinicians should advise against device use before bed, as excessive smartphone exposure in the evening is associated with worsened sleep quality,[122] which, in turn, cyclically exacerbates daytime ocular fatigue.

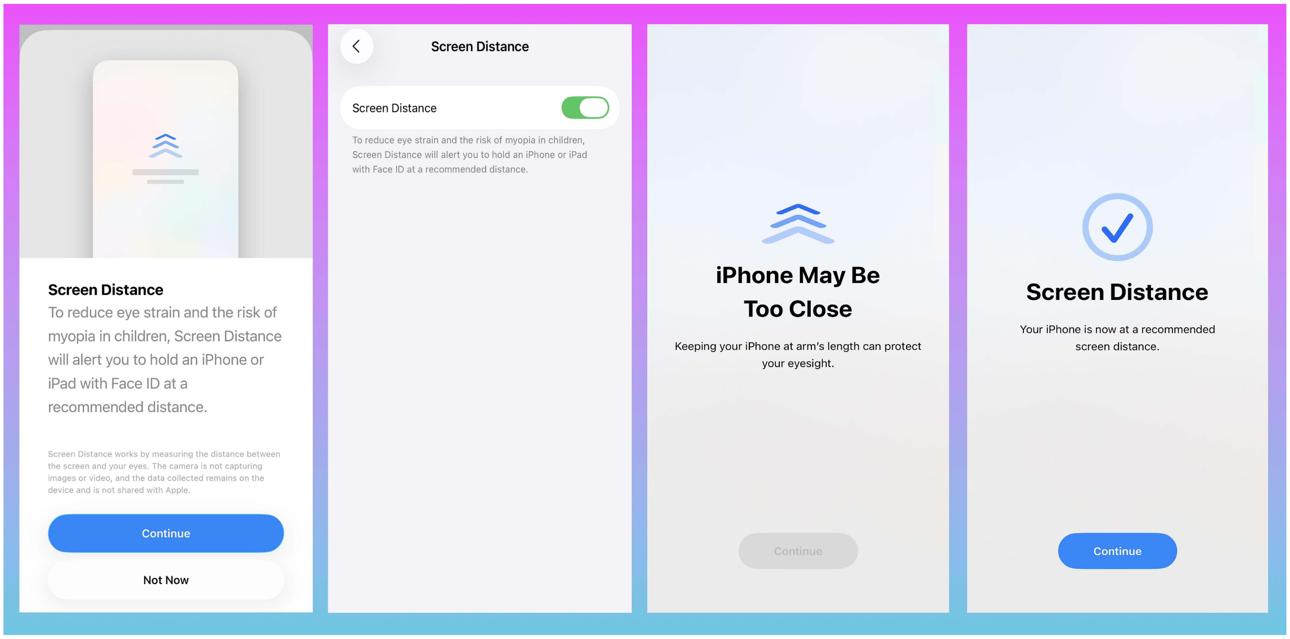
Furniture-related ergonomic interventions are essential for breaking the cycle of visual–postural strain. Ergonomic chairs with adjustable lumbar support reduce postural fatigue, minimizing the somatosensory drive that exacerbates discomfort.[133] Environmental control is equally critical. Maintaining thermal comfort (22–25°C) and relative humidity between 40% and 60% significantly stabilizes the pre-corneal tear film and reduces blink rate suppression.[77] Furthermore, ambient lighting should be optimized to prevent glare, with guidelines recommending lower ambient illumination (300–500 lux) for modern LED/LCD displays to maximize contrast without inducing disability glare.[133]

Pediatric Considerations

For the management of children, addressing the progression of refractive error is necessary. Screening for eye strain and refractive error through targeted school-based interventions has been shown to significantly improve visual acuity and reduce symptoms.[112]

Device Characteristics and Ergonomics

Ergonomic interventions should be specific. The viewing distances of symptomatic users are often closer, approximately 30 cm for phones, than those of clinical normals.[69] Recently, integrated biofeedback tools (such as the "Screen Distance" feature on iOS) have utilized TrueDepth camera metrics to provide real-time occlusion alerts when viewing distances fall below 30 cm (Figure 4). This effectively automates the maintenance of a physiological working distance, converting a passive recommendation into an active behavioral constraint that reduces accommodative load.[140] Interventions that increase viewing distance or use ergonomic display options, such as larger, curved monitors (eg 49 inches), have been shown to reduce trapezius load more effectively than dual-monitor setups.[75] Behavioral strategies, such as the 20-20-20



To reduce eye strain and the risk of myopia in children, Screen Distance will alert you to hold an iPhone or iPad with Face ID at a recommended distance.

Screen Distance works by measuring the distance between the screen and your eyes. The camera is not capturing images or video, and the data collected remains on the device and is not shared with Apple.

To reduce eye strain and the risk of myopia in children, Screen Distance will alert you to hold an iPhone or iPad with Face ID at a recommended distance.

Keeping your iPhone at arm's length can protect your eyesight.

Your iPhone is now at a recommended screen distance.

Figure 4 Apple's screen distance feature for digital eye health. Screenshots illustrate Apple's Screen Distance tool, which uses device sensors to monitor viewing distance and alert users when the iPhone or iPad is held too close. This is designed to reduce eye strain and myopia risk—especially in children—the feature provides real-time feedback and encourages healthier screen habits without capturing or sharing visual data.

rule, are widely recommended. Although recent evidence suggests that frequent, self-paced micro breaks may be more effective at stabilizing accommodation than single, scheduled breaks.[94]

Spectral Tuning and Blue Light

Recommendations regarding blue light require nuance owing to conflicting evidence. While multicolor filters have been shown to reduce beta-wave markers of cortical arousal,[109] and blue-blocking lenses may preserve contrast sensitivity under bright LED exposure,[86] a recent double-masked, controlled trial found that blue-filtering lenses provided no measurable benefit for dry eye signs or symptoms during a 120-minute reading task compared with standard antireflective coatings.[89] Therefore, spectral tuning should be prioritized for circadian regulation or specific contrast needs rather than as a blanket treatment for dry eye symptoms.

Discussion

The evidence synthesized in this review supports the hypothesis that digital eye strain is not a singular ocular condition but a complex, cross-system syndrome in which cognitive load, ocular physiology, and musculoskeletal mechanics interact.[48] Unlike traditional methods that isolate dry eye or refractive error, the literature reveals a cyclic "neuro-ocular" pathway in which central cognitive demands compromise peripheral physiology, which in turn feeds back to amplify central fatigue.[17,27] The "cognitive override" of the blink reflex surfaces to be a primary driver of digital eye strain. While screen use reduces blink frequency, studies have confirmed that cognitive demand, independent of the display medium, is the critical modulator.[2,26] A high cognitive load not only suppresses blink rate but also significantly increases the proportion of incomplete blinks.[6,7] This creates a specific mechanism for symptom generation: cognitive intensity leads to ocular surface exposure, which degrades the optical quality of the tear film. As tear film stability (NIBUT) decreases, it destabilizes the retinal image, forcing the accommodative system to constantly micro-adjust, thereby converting a surface problem into an internal neuromuscular burden.[11,68]

This review also clarifies the biological link between "eye strain" and "neck pain", problems that most clinicians treat as separate ergonomic concerns. Zetterberg et al and Richter et al demonstrated that this link is physiological rather than postural.[56,70] The incongruence between accommodation and vergence demands—or even pure ciliary muscle contraction at close distances—triggers a co-activation of the trapezius muscle.[56,57] Uncorrected refractive error or binocular dysfunctions are direct contributors to musculoskeletal rigidity. Consequently, optometric interventions that relieve accommodative-vergence conflict may have therapeutic value for neck and shoulder tension, positioning the optometrist as a key player in occupational musculoskeletal health.[138,141]

A recurring limitation in digital eye strain management is the reliance on patient-reported symptoms, which often lag behind physiological fatigue.[96] The reviewed studies highlight the utility of objective biomarkers. Accommodative microfluctuations, particularly high-frequency microfluctuations, have emerged as robust markers of ciliary muscle stress and cognitive load.[11,60] Increased frontal theta and reduced occipital alpha power in EEGs indicate cortical fatigue, which subjective questionnaires may overlook.[11,60] For the clinician, this finding supports integrating objective metrics, such as noninvasive tear breakup time (NIBUT) and standardized phoria testing, which correlate better with functional decline than symptoms alone.[42,55]

The role of blue light in digital eye strain is nuanced. Although evidence confirms that evening exposure to blue-enriched LED lighting strongly suppresses melatonin and disrupts sleep in both children and adults,[111,142] its daytime effects are distinct. Blue light enhances cortical efficiency and sustains attention. However, short-wavelength light scatters and reduces contrast sensitivity, and spectral tuning that reduces blue output also lowers dry eye symptoms in patients.[84,111] Thus, management should not be a comprehensive recommendation to "block blue light", but rather a context-specific strategy prioritizing spectral comfort and contrast protection during the day, and strict blue-depletion in the evening to protect circadian physiology.

The multifactorial nature of digital eye strain necessitates a staged management approach. Primary care must move beyond "lubricant eye drops and breaks" to address the specific driver. For children, the strong link between smartphone use and myopic progression mandates behavioral limits and outdoor activity.[113,114] For presbyopes, low-power convex adds (+0.75 D) have shown apparent efficacy in improving reading speed and comfort.[130] Finally, for patients with persistent symptoms, addressing "invisible" drivers—such as flicker sensitivity and binocular incongruence—is essential.[65,79]

This review has several strengths and limitations, including the integration of diverse study designs to form a comprehensive mechanistic framework. However, the review must acknowledge certain limitations. First, because a single author conducted this review, the study cannot rule out the potential for selection or appraisal bias. However, strict adherence to the PRISMA guidelines and the MMAT operational manual must mitigate this risk. Second, the included studies exhibited significant heterogeneity in the definition of "digital eye strain"—ranging from symptom surveys to objective critical fusion frequency tests—which precluded a quantitative meta-analysis. Third, the study limited the interpretation of these findings to the study populations, which were predominantly young, healthy adults or university students. Finally, most reviewed studies used relatively short exposure durations (less than 60 minutes), which may fail to capture the growing fatigue that accumulates over a typical workday.[135] The trajectory of digital eye strain in presbyopic populations and children requires longitudinal investigation, particularly to determine how early and cumulative digital exposure influences refractive development. Future research should prioritize real-world monitoring with wearable sensors (mobile EEG, eye-tracking, and ocular surface sensors) to validate laboratory findings in naturalistic work and educational environments.

Conclusion

Digital eye strain is a collision between evolved visual physiology and modern technology, in which the visual system's efforts to maintain clear vision under sustained cognitive and environmental stress lead to systemic fatigue. The evidence supports a shift from generic advice to a mechanism-based management algorithm: identify the dominant driver—ocular surface instability, binocular dysregulation, or device-related optical stress—and apply targeted interventions (for example, blink training and ocular surface care; refractive/binocular correction and vision therapy; ergonomic and display optimization). Such tailored care can interrupt the neuro-ocular feedback loop and improve symptoms and function. Future work should validate these staged pathways in longitudinal, real-world studies and standardize objective biomarkers to enable personalized, evidence-based care.

Data Sharing Statement

All data generated and analyzed during this study are included in the article and its supplementary files.

Funding

There is no funding to report.

Disclosure

The author reports no conflicts of interest in this work.

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